

REPRODUCTIVE SYSTEM OF WOMEN WITH AUTOIMMUNE THYROIDITIS

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Abstract

The work examines the causes and provoking factors of the occurrence of autoimmune thyroiditis, which are associated with the human hormonal system. It is shown that cervical dysbacteriosis changes the immune response, which leads not only to high exposure to antigens, but also to local inflammation.

Keywords: Thyroiditis, inflammation, intestine, hormone, manifestation, subclinical hypothyroidism.

Introduction

Today, great importance is attached to the problem of the demographic crisis, which is only gaining momentum and can lead to unfavorable consequences.

This situation is caused by a number of reasons. On the other hand, the health of men and women is deteriorating due to unfavorable environmental conditions and changes in attitudes toward their own health. The majority of the younger generation prioritizes their careers, their own desires and interests, so many women are in no hurry to start a family and have children and resort to terminating an unplanned pregnancy [1]. Along with the above problems, there are cases when families, on the contrary, want to have children, but women's attempts to get pregnant and carry a pregnancy to term are unsuccessful.

In this case, we are talking about infertility or failure to carry a pregnancy to term, which can be caused by many diseases of the endocrine system, in particular, autoimmune diseases of the thyroid gland [2]

Given the widespread prevalence of thyroid diseases in the population and their impact on reproductive function, this problem seems extremely relevant. At the same time, in a significant number of cases, the tactics of managing patients with autoimmune thyroiditis are limited to observation, including women suffering from infertility and miscarriage, and having no other obvious causes of reproductive disorders.

Various researchers have already proven that the presence of thyroid tissue AT does not coincide with the presence of either manifest or subclinical hypothyroidism: that is, AT can be detected even in individuals who do not have any changes in the functioning or structure of the thyroid gland [3]. On the other hand, women with elevated levels of thyroid AT, even in a euthyroid state, have a significantly higher frequency of complications during pregnancy and

childbirth; and the administration of L-thyroxine in this situation does not reduce the risk of these complications [3].

Autoimmune thyroiditis (AIT) is a chronic disease in which cells are gradually destroyed. The thyroid gland's functionality is impaired, as it is affected by autoantibodies. Therefore, the disease is defined as autoimmune. The causes of AIT are not fully understood, but specialists have found a way to significantly slow down the development of the pathology. Let's take a closer look at what kind of disease it is, why it develops, what symptoms it has and how to treat it.

AIT is often called Hashimoto's disease. This is the name of the scientist who was the first to describe the pathology and identify the factors that provoke its development. This is a disease in which the human immune system begins to destroy the structure of the thyroid gland, destroying cells.

Often this leads to hypothyroidism - a certain condition in which the thyroid gland stops producing the required amount of hormones. Because of this, the cardiovascular system and metabolic processes may suffer.

Tissue inflammation becomes chronic as a result of autoimmune changes. It is impossible to cure AIT completely, but doctors help eliminate the symptoms, remove the consequences of hormone deficiency and protect the body from damage.

Autoimmune thyroiditis has several forms of progression, here are the main ones:

- Chronic AIT. Occurs due to increased growth of T-lymphocytes. Hypothyroidism develops against the background of this autoimmune pathology. The most common form of the disease.
- Postpartum thyroiditis. Also very common, appears due to changes in the structure of the woman's body during pregnancy and hormonal surges. It is enough to simply recover, the function of the gland stabilizes.
- Painless form. The most unpredictable type, it is difficult to understand the course of the disease, it is difficult to notice hormonal disorders at the first stage. For detection, it is recommended to undergo a periodic comprehensive examination.
- Cytokine-induced thyroiditis. Occurs due to the use of pharmacological drugs in liver damage, blood disorders. The treatment process is long and complex.

Modern diagnostic methods allow you to accurately determine the form of the disease, the level of hormones and the condition of the thyroid gland. After that, the doctor prescribes treatment depending on the form. In most cases, AIT can be cured quite successfully. It is impossible to get rid of it completely, but it is easy to prevent further development. The main thing is to seek help from specialists in a timely manner.

The peculiarity of the disease is that the symptoms change depending on the amount of a certain type of hormone. The disease can generally proceed without any symptoms, not causing pain or discomfort. Sometimes it is discovered by chance, during an examination or routine testing. But certain symptoms are often present. The symptoms of autoimmune thyroiditis are as follows:

- lethargy of the body occurs, accompanied by apathy and a depressive state;
- weight can be rapidly gained or, conversely, lost for no reason;

- hair loss;
- the skin becomes dry;
- a sore throat occurs regularly;
- the voice changes, becomes hoarse and rough;
- memory deteriorates;
- the thyroid gland increases in size;
- problems arise in the functioning of the heart;
- the heartbeat becomes more rapid, tachycardia develops;
- tremors of the limbs appear;
- irritability and severe fatigue arise, which cannot be gotten rid of even after a long sleep;
- mood changes regularly.

If you suffer from such symptoms, you should contact a medical center and undergo diagnostics. All this may indicate the presence of an autoimmune disease. The sooner you take measures to restore the hormonal system, the better the result will be.

Experts cannot determine the final causes of autoimmune thyroiditis. The human hormonal system has not been fully studied, so autoimmune diseases are quite difficult to treat. But doctors identify provoking factors that contribute to the development of thyroiditis :

- inflammatory processes in the thyroid tissue;
- the presence of chronic infections throughout the body;
- uncontrolled use of various medications;
- consuming large amounts of iodine in any form;
- regular stress;
- irradiation;
- ARI, flu condition;
- pregnancy and childbirth, during which the likelihood of developing thyroiditis increases by 20%;
- genetic factor.

Genetic changes and predisposition are considered the main cause of the development of this disease. Doctors' opinions on this issue differ. The problem is that it is impossible to determine any of the reasons that clearly provoke disorders in the thyroid tissue. Therefore, predisposition is defined as the main factor. If there are relatives in the family suffering from thyroid diseases, special attention should be paid to this issue.

To eliminate the consequences of the disease and prevent its further development, it is necessary to choose an effective course of health improvement for an autoimmune disease. It is important to conduct a detailed analysis of the thyroid gland. This will allow you to accurately determine which hormones should be given attention and how to monitor the patient's condition.

As a rule, the diagnosis is made on the basis of clinical manifestations, detection of thyroid tissue pathologies and a number of tests. The tests allow determining the form of the autoimmune disease, the degree of development of the pathology. After receiving the results, the specialist prescribes treatment, choosing the most optimal methods of influence.

Impaired reproductive function in women with autoimmune thyroiditis Violation of reproductive function in females with autoimmune thyroiditis fertilization and pregnancy [7, 8].

Therefore, it is quite obvious that a high prevalence of infertility (almost 50%) among women with thyroiditis has long been shown. Hashimoto's (chronic lymphocytic thyroiditis) or Graves' disease .

Both of these diseases include hyper- and hypothyroidism, disrupt the processes of folliculogenesis , affect the metabolism of sex hormones and contribute to the formation of anovulatory infertility, and during pregnancy, increase increase the frequency of early pregnancy losses and negatively affect the fetus and the health of newborns [6, 7].

If the hypothyroidism condition is observed over a long period, then the secretion of thyrotropin - releasing hormone increases in the hypothalamus, the function of which is to stimulate the secretory function of the thyroid gland. Consequently, the level of thyroid-stimulating hormone (TSH) and prolactin increases significantly, against which background the probability of developing secondary hyperprolactinemia , galactorrhea , anovulation and menstrual cycle disorders increases [5].

An important role in the development of infertility with changes in thyroid function is played by sex hormone binding globulin (SHBG), which is produced by the liver. Its role is to bind and transport testosterone, estradiol and 5- α - dihydrotestosterone , as well as to protect against metabolic inactivation along the way from the site where they are synthesized to the target organ [5].

The thyroid gland is also responsible for the synthesis of SHBG, stimulating its production in optimal quantities. However, with hypothyroidism, there is a lack of SHBG production, an increase in the concentration of free fractions of estrogens and androgens, as a result of which there is a violation of the mechanism of positive and negative feedback with the hypothalamic-pituitary system, which is manifested by menstrual cycle disorders and a decrease in reproductive function due to anovulation [5].

With the development of hypothyroidism, there is also a decrease in the rate of metabolic clearance of androstenedione and estrone, and the degree of their peripheral aromatization increases [5]. Therefore, during preconception preparation for pregnancy, there are several points that must be taken into account: the presence of thyroid dysfunction in autoimmune thyroiditis with the formation of hypothyroidism or thyrotoxicosis - on the one hand, and the presence in the female body of autoantibodies to thyroid tissue without changing its function, on the other.

The prevalence of autoimmune thyroiditis (AIT) is not precisely known, due to the fact that in the euthyroid phase of AIT there are no precise diagnostic criteria. However, the frequency of detection of antibodies to thyroid tissue in women of reproductive age (TG), for example, the carriage of antibodies to thyroid peroxidase (AT-TPO) is about 10% and depends on the ethnic composition of the population [1-6].

Sex hormones appear to be linked to the immune response, leading to a higher incidence of autoimmune diseases, a common autoimmune disease that affects between 5 and 20% of women of reproductive age. Women have an 8-fold higher risk of AIT than men [7].

There is compelling evidence that environmental agents play a critical role in triggering autoimmune diseases in genetically susceptible individuals. In particular, exposure to polyhalogenated Biphenyls, which are widely used in industry, such as in paints and adhesives, may increase levels of antimicrosomal thyroid antibodies and thyroglobulin antibodies.

The development of a number of autoimmune diseases, including AIT, can be facilitated by exposure to crystalline silica (a mineral widely used in construction, radio engineering, and the aviation industry).

A large number of common environmental pollutants can activate epigenetic modifications that play an important role in the development of thyroid diseases. Vitamin D deficiency has also been suggested as a predisposing factor for autoimmune diseases and has been shown to be reduced in patients with thyroid autoimmunity.

In turn, its deficiency is also associated with infertility and miscarriage, suggesting a potential interaction with thyroid autoimmunity in the context of infertility. Evidence is gradually accumulating about the complex relationship between the thyroid gland and the intestine.

Recent studies demonstrate an important correlation regarding the influence of intestinal bacteria on the immune system and thyroid function. Intestinal dysbiosis alters the immune response, promoting inflammation and reducing immune tolerance, and as a result leads to damage to the intestinal mucosa and increased permeability, respectively, leading not only to high exposure to antigens, but also to local inflammation.

Also, intestinal dysbacteriosis can directly affect the level of thyroid hormones due to its own deiodinase activity and inhibition of TSH (8-17).

Pregnancy itself is the creation of a unique bond between mother and fetus. Half of the fetus's DNA is inherited from the father, and therefore is an allogeneic substance for the mother. However, the maternal immune system does not reject the semi-allogeneic fetus during normal pregnancy - maternal immune tolerance is ensured by the predominance of T-helper 2 (Th 2) cytokines, the immunosuppressive function of regulatory T cells (Treg) and subtle immune with cross-linked changes at the interface between mother and fetus.

A review of the literature has shown that in women with AIT, proinflammatory Th 1 immune responses and excessive activation of B cells, NK cells, and NKT-like cells may impair maternal immune tolerance, leading to miscarriage. pregnancy; (2) Women with AIT may have concomitant ovarian autoantibodies, and the presence of thyroid autoantibodies and proinflammatory cytokines may affect oocyte immune homeostasis, which may impair ovarian function. reserve, oocyte fertilization and embryogenesis; (3) In mice with thyroiditis Hashimoto's suppressed expression of receptivity markers (ICAM-1, LIF and integrin $\beta 3$) indicates decreased endometrial receptivity, which may affect the implantation process.

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